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MUSCULAR CONTRACTION

A

PHYSICAL PHENOMENON.

A SUMMARY OF THE ARGUMENT

WITH

ALTERATIONS AND ADDITIONS.

BY

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MUSCULAR CONTRACTION,

ETC.

In the following pages it is proposed to give a summary of the principal arguments which have been advanced at different times,* and which seem to show the necessity of a complete revolution in the opinions at present held respecting muscular contraction. This summary is divided into four parts. In the first part, it is proposed to show that muscle is not stimulated to contract by any of the commonly reputed stimuli,—electricity, nervous influence, blood, light, heat, and the rest. In the second part, it is proposed to examine into the nature of muscular contraction, and point out reasons for supposing that this contraction is nothing more than a passive physical consequence of the molecular attraction of the muscle. In the third part, it is proposed to show that the special muscular movements which are concerned in carrying on the circulation—"capillary motion" and the rhythm of the heart—can only be explained upon these views of muscular contraction. In the fourth part, it is proposed to glance at the pathology of muscular contraction, and show that this is in harmony with the physiological premises.

I.

In this part, the object is to show that muscle is not stimulated to contract by any of the commonly reputed stimuli,—electricity, nervous influence, blood, heat, and the rest.

1. *Of Muscular Contraction in relation to Electricity.*—Living muscle is the seat of electrical currents which proceed from the ends to the sides of the fibres, and which may be traced, not only in the entire muscle, but in the individual fibres. These currents continue during life, and are not finally extinguished until the oc-

* These views were first propounded about five years ago, in a work called 'The Philosophy of Vital Motion;' and since this time they have been elucidated in various ways, particularly in a book having for its title, 'Epilepsy, and other Affections of the Nervous System which are marked by tremor, convulsion, or spasm: their Pathology and Treatment.' (Churchill, 8vo, 1854.)

currence of *rigor mortis*. They are designated under the general name of "muscular current," and they will be so designated here.

The muscular current continues during life, but it does not always continue at the same pitch of intensity. It changes during muscular contraction. Matteucci, who first directed attention to this subject, thought it became stronger at this time. Du Bois-Reymond,* on the contrary, first thought it became weaker, and then doubted his original conclusion. The facts, however, are sufficiently simple, and there need be no doubt as to their significancy.

If a living muscle be connected in a proper manner with the galvanometer, the needle immediately reveals the muscular current by diverging to a certain distance from zero; and this it does until the occurrence of contraction, when it immediately moves to the other side of zero. The needle, that is to say, is acted upon by a *reverse* current during contraction. There is no doubt about this, but there is doubt about the origin of the current. Is it in the muscle or in the instrument, for it may be in either or both? This question is answered by testing separately the condition of the muscular current during rest and during contraction (by breaking the connection and depolarizing the galvanometer between the two experiments); and the answer is, that the needle moves in the *same* direction during contraction as it moved during rest, but not to the same distance from zero. In other words, the muscular current is *weakened during contraction*, but not changed; and hence it follows that the backward movement of the needle during contraction in the former experiment was due to the secondary reverse current which traverses the coil of the galvanometer upon the diminution of the current which had previously passed from the muscle through the coil.

Judging from the needle of the galvanometer, then, the muscular current appears to be *weakened during contraction*, and why then does Du Bois-Reymond doubt a conclusion which appears to be so obvious? He doubts, because the phenomenon of "secondary contraction" (which is induced in a second muscle by placing its nerve in a certain manner upon the muscle which is being made the subject of experiment) appears to reveal the presence of certain *changes* in the muscular current of the primarily contracting muscle which are not revealed by the needle of the galvanometer. Now, certainly, the "secondary contractions" are due to certain *changes* in the muscular current of the primarily contracting muscle. All are agreed about this. But what is the nature of the change? Du Bois-Reymond argues that the "secondary contractions" could not arise if the muscular current (which causes them) remained *constant*, and he therefore concludes that there must be certain *oscillations* in the current during contraction, although the sluggish needle of the galvanometer gives no evidence of them. But this is begging the whole question, as will be seen presently.

The commonly received opinion respecting the action of artificial electricity upon muscular contraction is that there is no contraction so long as the current is constant, and that contraction immediately

* 'Untersuchungen über Thierische Electricität,' Berlin, 1848.

follows whenever the current is found to rise or fall in any manner. Is it so? What are the facts, and is this their significance?

When an artificial current is passed through the leg of a frog in a direction opposed to that of the muscular current of the limb, there is a *strong* contraction on making, and a *slight* contraction on breaking the circuit, and there is *no* contraction during the passage of the current. In other words, there is a *strong* contraction at the moment when the artificial current neutralizes and is neutralized by the muscular current; there is *no* contraction when the artificial current prevails and is passing through the muscle; and there is a *slight* contraction when the artificial current is suspended. This latter contraction is less marked than the other, and this may well be, for the slight reverse current, which must be set up in the muscle on the suspension of the primary artificial current, must pass in the same direction as the muscular current; and because this reverse current is too feeble to interrupt the re-establishment of the muscular current, the muscular current may resume its action with such rapidity that there is no time for any marked degree of contraction. In a word, the simple fact appears to be that contraction is absent when electricity is manifestly present, and present when electricity is in all probability absent.

When, on the other hand, the artificial current is passed through the leg of a frog in the same direction as the muscular current, there is a *slight* contraction on making, and a *strong* contraction on breaking, the circuit, and there is *no* contraction during the passage of the current. There is still *no* contraction while the muscle is acted upon by the full force of the artificial current; and there is a *strong* contraction when the artificial current is suspended, and when the reverse current which is set up on this suspension must, for the time (the two currents being contrary), neutralize and be neutralized by the muscular current, which muscular current recovers itself when the artificial current is suspended. But what of the *slight* contraction which occurs on making the circuit,—when, that is to say, the artificial and natural currents coincide, and when, apparently, they ought to intensify each other? This is, indeed, a difficulty, but the explanation appears to be that the two currents do not intensify each other, although they coincide in their direction; but that the artificial current, being the stronger, suspends the muscular current, by altering for the time that definite molecular arrangement upon which the muscular current depends. In this case, therefore, the contraction is coincident with the moment of suspension, the contraction being slight because the artificial current establishes itself without having to encounter any direct opposition from the muscular current. In this experiment, then, as in the other, there is no sufficient reason for referring the contraction of the muscle to the stimulus of electricity, and the rule still appears to be that contraction is absent when electricity is present, and present when electricity is absent.

It is the same also if the artificial current be passed across the muscular current, for the contraction still happens on making and breaking the circuit, and not during the passage of the current. There is *no* contraction, that is to say, while the muscle is acted upon by the

artificial current; there is contraction before the artificial current is established, and at the moment when this current is conflicting with the muscular current; and there is contraction before the muscular current is re-established, and at the moment when the returning muscular current is conflicting with the reverse artificial current.

The fact, however, which is more conclusive than any other as to the influence of artificial electricity upon muscular contraction has just been furnished by M. Eckardt.* *It is that a tetanized muscle is relaxed by the passage of a constant current of electricity.* This fact is, indeed, an *experimentum crucis*, when taken in connection with the facts already cited. These have shown that contraction is absent when electricity is present in the muscle; this shows that contraction must cease if electricity be communicated to the muscle.

It would thus appear that the opinions generally held respecting the action of electricity upon muscle are not tenable, and that it is not enough to say that there is no contraction so long as the current is constant, and that contraction immediately follows whenever the current rises or falls in any manner. On the contrary, the simple facts appear to be that contraction is absent when the natural or artificial currents are present, and that contraction is only present when the muscular current is in some degree absent. Nay, so antagonistic is the current to contraction, that it even dispels it when present. And if this be the case, then the conclusion which Du Bois-Reymond has drawn from the phenomenon of "secondary contraction" falls to the ground, for all that is necessary to the production of this phenomenon is, not that there should be *oscillation* in the muscular current of the primarily contracting muscle, but simply that this current should become *weakened;* and thus the position of the needle of the galvanometer and the phenomenon of "secondary contraction" alike point to the same fact, and that is a *fall in the muscular current during contraction.*

— In no point of view, then, does muscle appear to be stimulated into contraction by electricity. On the contrary, the only safe conclusion which can be drawn from this intricate evidence is altogether opposed to this idea. It is that muscular contraction is antagonized by electricity.

2. *Of Muscular Contraction in relation to Nervous Influence.*—Comparing involuntary with voluntary muscles, the involuntary muscles are found to be most disposed to contract. They contract less readily and energetically, but when they do contract the contraction is more abiding. But these very involuntary muscles are the muscles which are less liberally supplied with nerves, and hence the disposition to contract appears to be inversely related to the supply of nervous influence. At any rate this was the inference which Hunter drew from the fact; for he says, "the voluntary and involuntary muscles, having their quantity of motion in an inverted proportion to their quantity of nerves, is a strong argument against the nerves being the cause of muscular motion."†

* 'Grundzüge der Physiologie des Nervensystems,' Giessen, 1854.

† 'Hunter's Works,' by Palmer, vol. iv, p. 213.

Nor is this inference unsupported by other facts.

The first of these is furnished by M. Eckardt in an experiment in which he tests the influence of heat upon the "irritability" of the nerve. In this experiment, the prepared leg of a frog, with a large portion of its nerve attached, is immersed in water at various degrees of temperature. At the natural heat of the animal—about 30° Reaumur—the "irritability" of the nerve is not appreciably affected, for, on touching the nerve with a needle, the muscle contracts as readily as it did before it was put into the water. As the temperature rises, however, the "irritability" progressively diminishes; and when the thermometer stands at 54° Reaumur, or thereabouts, it is no longer possible to provoke contraction by touching the nerve. Now the point of interest to the question at issue is this, that the muscle contracts in obedience to the action of the heat, when the temperature is sufficiently high to destroy the "irritability" of the nerve. In other words, the muscle is exhibited as contracting coincidentally with the loss of "irritability" in the nerve; or, as M. Eckardt expresses it, "das Zustandekommen der Zuckung durch eine momentane Zerstörung des Nerven bedingt sei."

A fact of similar significancy is to be found in the changes which Du Bois-Reymond has shown to take place in the electrical currents of nerves, for these currents (which may be designated hereafter as the *nerve current*) are found to become weakened during contraction. They behave precisely like the muscular current. Is this, then, an argument that the nervous influence is similarly diminished at this time?

A third fact is one which seems to show that the nervous centres induce contraction by suspending the nerve current. The fact is this. If a frog be thrown into a state of tetanus, its nerve currents are found to be weakened wherever they are examined; but if a nerve be divided, the tetanus immediately ceases, and the nerve current returns in the parts below the section. What then? Is it not the inference that the nervous centre had induced the tetanus by suspending the nerve current? and, if so, is there not a collateral inference that nervous influence had been suspended at the same time?

But what is nervous influence? Is it a distinct agency, or is it (so far as the muscles are concerned) the influence exercised by the nervous centres in and through the electrical currents of which nerves and muscles are undoubtedly the seat? Is it that living *quiescent* centres, nerves, and muscles, are all the seat of these currents, and that muscular contraction is brought about by the suspension of these currents? Is it that *plus* becoming *minus* in the nervous centre (be this by an act of the will or by other means), a similar change from *plus* into *minus* takes place by *conduction*, first in the nerve and then in the muscle, and that contraction is the effect of this change in the latter organ. According to this view nervous influence is presented to the mind rather as a process than as an agency, and the two facts last mentioned become direct instead of indirect illustrations of what takes place in the nerves during muscular contraction. Be this as it may, however, and be the nervous influence an agency or a process, the presumption which arises out of the facts already mentioned

is, that the influence in muscular contraction is one of e-nervation and not one of in-nervation.

But if muscular contraction holds this relation to nervous influence, ought not the muscles to be contracted when the muscle is cut off from the nervous centre by dividing or otherwise paralysing the nerve, or when the action of the nervous centre is diminished, as during sleep? Is not the fact that the muscles are generally relaxed under these circumstances a proof that the nervous centres communicate something to the muscles which is necessary to contraction? By no means.

In explaining these apparent objections it is only necessary to remember the law of the nerve current and muscular current.

It is necessary to remember that the nerve current is always present in living nerve during quiescence; that this current is weakened when the nerve is occupied in producing contraction; and that it is restored to its former degree of power when the nerve has ceased to cause contraction.

It is necessary to remember that the muscular current is always present in living muscle during quiescence; that this current is weakened during contraction; and that it is restored to its former activity immediately after contraction.

It is also necessary to remember that these particular currents are inherent in nerve and muscle, and that they continue to be manifested in detached fragments of nerve and muscle for some time after they are removed from the body. They are, no doubt, weakened under these circumstances, but their presence is unmistakeable.

These are the fundamental facts which afford the clue to the solution of the difficulties alluded to, and of many others which remain behind. It does not follow, then, that a muscle ought to remain contracted when it is cut off from the nervous centre by dividing or otherwise paralysing the nerve. On the contrary, there are no natural means of suspending the muscular current now that the nerve is paralysed; and, according to the premises, therefore, the muscular current, which is always present when the muscle is left to itself, will always keep the muscle in the relaxed state. This muscle may, and generally does, contract at the instant of paralysis, because its current may then be weakened by being cut off from the currents which play in the nerves, and, probably, also in the nervous centres. It may contract momentarily under the touch of a foreign body, as will be explained presently. It may contract permanently when the paralysis has continued some time, and when the polar and nutritive action of the muscles have both failed, as in those long standing cases of paralysis which are so well described by Dr. Todd. It will contract permanently in *rigor mortis*, when the muscular current has ceased altogether. But a healthy living muscle does not remain contracted when the nerve is divided or paralysed in any other way, and it ought not to remain contracted because the muscular current continues, and because there are no *natural* means of suspending this current now that the muscle is cut off from the nervous centres.

And if the muscles ought not to remain contracted, in cases where

the influence of the nervous centres is cut off by paralysis of the nerve, they ought not to remain contracted in sleep, where the influence of these centres is only diminished. At the moment of falling asleep, there are usually, if not invariably, some muscular startings, which may show that some nervous influence has been cut off from the muscles; but these startings once over, the nervous centres, the nerves, and the muscles resume their polar play; and, though their action is probably less energetic than when the nervous centres were in the waking state, it may be assumed to be more than sufficient to counteract any very appreciable degree of muscular contraction, seeing that the action which continues in a muscle for some time after its removal from the body is sufficient to do this. Muscular contraction may indeed occur during sleep, but if it does do so, there has been some additional suspension of the action of the brain, or other nervous centre, either through the influence of the will in a dream, or by some unusual failure of the circulation, such as generally operates when convulsion or spasm is brought about during sleep. But there is no reason whatever for supposing that the muscles should remain contracted during sleep.

On reviewing the whole evidence, therefore, there does not appear to be any good reason for believing that muscle is stimulated to contract by nervous influence, and there is much evidence to the contrary.

3. *Of Muscular Contraction in relation to the Blood.*—It is equally difficult to suppose that muscular contraction is in any way caused by the blood. The tendency to prolonged contraction appears to be inversely related to the supply of blood; thus this tendency is greater in the voluntary muscles of fishes and reptiles than of mammals and birds, greater in involuntary than in voluntary muscles, and greater in the muscles of any given animal during the state of hybernation than during the period of summer life. The fact, also, that the state of *rigor mortis* may be relaxed more than once, and the lost "irritability" restored to the muscle, by the injection of living blood into the vessels,—a fact which has been recently and repeatedly verified by M. Brown-Séguard,—appears to be in direct contradiction to the idea that muscular contraction is stimulated by the blood.

Nor is it necessary to have recourse to the contradictory doctrine, that "the degree of irritability is inversely related to the rate of respiration," in order to account for the first-named of these phenomena. On the contrary, it is only necessary to suppose that the force of the muscular current is in direct relation to the supply of blood, and that the contraction is resolved, by the re-establishment of this current, with a rapidity which bears a direct relation to the supply of blood; and then the more marked disposition to contract, when the supply of arterial blood is deficient, means nothing more than that the muscular current, and the attendant relaxation of the fibre, are re-established with greater slowness.

There are, however, sundry facts which seem opposed to the idea that muscular action is antagonized by the blood.

In hemorrhage, an animal is convulsed when its state verges upon syncope, and the convulsion seems to depend upon loss

of blood; but, when its state is one of actual syncope, the convulsion passes off, and the muscles remain relaxed until the occurrence of *rigor mortis*. It seems as if the convulsion requires the stimulus of a certain amount of blood.

In asphyxia there is a similar order of phenomena. In this state the involuntary muscles are first affected, and the intestines writhe about like so many snakes; then the convulsions become general; but when the blood has entirely lost its arterial properties, and the asphyxia is complete, the convulsions cease, and the muscles are perfectly relaxed. It still seems as if the stimulus of the aerated blood is necessary to the convulsion.

In death, also, the convulsion of the agony ceases when death gains the mastery, and the muscles remain relaxed until the occurrence of rigor mortis; and so it might be expected, for, as far as the circulation is concerned, death is only syncope or asphyxia, in which there is no rallying.

In all these cases, however, there is a fallacy, and in reality they afford no manner of countenance to the idea that muscular contraction is stimulated by the blood. The facts remain, but not the interpretation which has been put upon them.

When the muscles cease to be convulsed in syncope, asphyxia, or death, this cessation is certainly not due to loss of contractile power in the muscles, for these very muscles contract vigorously under the influence of galvanism, or when pricked with a needle, and last of all they contract firmly and entirely in rigor mortis. What is lost is the faculty of responding to certain changes in the nervous centres. When the convulsion ceases, the simple fact appears to be, that the nerves have ceased to be conductors, and that the change from *plus* into *minus* in the action of the nervous centres (which is caused by hemorrhage on the one hand, and want of arterial blood on the other), is no longer *conducted* along the nerves to the muscles; and being no longer conducted, the muscular current is left at liberty to resume its play, and relax the muscular fibre. This interpretation is allowable, for the muscular current is far less dependent upon the supply of blood than the nerve current. Now, there is good reason for supposing that the nerves have ceased to be conductors in syncope, axphyxia, and death. When the circulation in the hand is suspended by immersion in iced water, the sense of touch and the power of movement are partially or wholly destroyed. When the principal vessel of a limb is tied, a similar result ensues, until the collateral circulation is established. In each case, also, the power of provoking reflex movements is diminished or destroyed. On the other hand, the sensibility, and the command over movement, are both increased when the circulation is roused by warmth, or in any other way. Facts such as these serve to show that the nerves require a certain supply of blood to enable them to act as conductors, and they warrant the conclusion that the nerves must have ceased to be conductors at the time when the convulsions of syncope, asphyxia, or death come to a termination, for at this time the supply of blood to the nerves is less than it is in the experiments in which the hand is plunged in iced water, or in which the principal vessel of a limb is tied,—a conclusion which is

collaterally supported by the fact, that the nerves, under these circumstances, have ceased to convey sensory and volitional impressions. And if so, then it is every whit as intelligible that the convulsions should cease, under these circumstances, as that a tetanized muscle should relax when the nerve is divided.

According to these premises, it is quite intelligible that convulsions should be the consequence of a state of circulation verging on syncope, and that convulsion should cease in syncope. In the state verging on syncope, the amount of blood passing through the vessels is insufficient to keep up the proper action of the nervous centres, but it is still sufficient to keep up some degree of conducting power in the nerves, and hence the convulsions; for, the nerves being conductors, that failure in the action of the nervous centres which is dependent upon the want of blood, conducted along the nerves, entails a corresponding failure in the muscular current, of which contraction or convulsion is the result. In actual syncope, on the other hand, the circulation is no longer sufficiently active to preserve the conducting powers of the nerves, and hence the cessation of the convulsions; for, the nerves being no longer conductors, the failure in the action of the nervous centres, however absolute, no longer involves a corresponding failure in the muscular current, and, not involving this, the current returns, and the convulsions are at an end.

The same train of reasoning applies to the case of asphyxia. So long as the blood is sufficiently aërated to preserve the conducting powers of the nerves, any failure in the action of the nervous centres, which is itself brought about by the want of arterial blood, may issue in convulsion; but when the nerves cease to be conductors—as they do when the blood has lost its arterial properties—then the muscular currents are no longer suspended by the suspension of the action of the nervous centre, however complete this suspension may be; and not being suspended, the muscular current resumes its sway, and the convulsion is done away with.

For the same reasons, the tremors, or convulsions, or cramps of the agony cannot continue after death: for if the nerves cease to be conductors in syncope and asphyxia, they must cease to be conductors when all circulation is at an end, and the blood stagnant.

According to the premises, therefore, it is quite intelligible that convulsion should appear in a state *tending to* syncope, asphyxia, and death, and yet cease *in* syncope, asphyxia, and death, and this without supposing for one moment that the immediate stimulus of blood is necessary to muscular contraction. Indeed, there is no one fact which can serve to show that muscle is stimulated to contract by the blood.

4. *Muscular Contraction in relation to various Mechanical Agents.*—Nor is it by any means probable that muscle is stimulated to contract by mechanical agents. Instead of exciting the bladder to contract, the urine accumulates, the viscus expands, and contraction seems to *happen* when further expansion is productive of uneasiness or pain. Instead of exciting the uterus to contract, the germ grows and the womb enlarges proportionately, and contraction, to all appearance, does not happen until the growth of the fœtus is perfected, and the

stimulus of that growth at an end. For nine long months the fœtus seems to have excited the uterus to continual expansion, and, to say the least, it is not easy to imagine how it can excite contraction at the time of labour. Arguing from the history of pregnancy, the probabilities, as measured by time, are those of nine months to as many hours against such a view. Discarding theory, indeed, the simple fact appears to be that the fœtus grows and causes the uterus to expand by the stimulus of its growing presence, and that it does this until that growth begins to trench upon the supplies which are necessary for the proper nourishment of the mother. Then the child becomes a source of exhaustion to the parent, and this exhaustion, reacting upon the uterus, brings back the state of contraction,—for, if the uterus expanded in consequence of stimulation, it must return to the state of contraction if the degree of stimulation be diminished, and this equally whether this diminution be caused by the death of the child, or by the child having lived so long that it begins to starve the mother by its too clamorous wants. In either case contraction must happen if the uterus had previously been kept in a state of expansion by stimulation. This contraction compresses the placentary vessels, and depresses the life of the fœtus by interfering with the proper aëration of the fœtal blood; and this depression, reacting upon the uterus, is attended by a further degree of contraction. This contraction, like the first, compresses the placentary vessels, and depresses the life of the fœtus by interfering with its respiration; and this depression, reacting on the uterus, necessitates a corresponding degree of contraction. Again and again, contraction leads to contraction by the same process, and in this way the uterus acts upon the fœtus, and the fœtus reacts upon the uterus, with ever-increasing contraction as the result, until the completion of birth. At all events, it is impossible, upon any rational view of parturition, to refer the contraction of the uterus to any *stimulation* on the part of the fœtus, without ignoring the whole previous history of pregnancy. Nor can it be successfully objected to this view that the bladder is excited to contract by a stone, or the uterus by the clots of blood which occasionally remain after delivery. There is no evidence whatever that the stone acts in this manner. The bladder is morbidly sensitive under these circumstances, and a very small quantity of urine is enough to cause distress or pain, and thus the *will* or *instincts* are roused to empty the bladder more frequently than usual. The uterus, also, goes on contracting after delivery, until the process be complete, and this equally whether there be clots in the cavity or not. If there are clots in the cavity, it only shows that more of the process of contraction remains to be effected than there ought to be; but it cannot show that the clots excite contraction, for in other cases the same contractions take place, and more effectually where no such clots are present.

It is not even certain that a needle stimulates contraction. The muscle does not always contract under these circumstances, and when it does, there is some reason to believe that the contraction may be due to the discharge of electricity previously present in the muscle. The fact that there is a disappearance of electricity at this time, and

the known analogy between the structure of the muscle and of the electrical organ of the torpedo, and between the circumstances attending the production of contraction on the one hand and of discharge on the other, are, to say the least, in favour of this supposition. But it may be objected that this contraction is provoked by the touch of a piece of glass or of any other non-conductor; and this objection is not easily disposed of. It may be, however, that that molecular arrangement of the muscle which is necessary to the existence of the muscular current is broken by the *pressure* of the touching body, in which case there would be a loss of action similar to that which would happen in a galvanic pile, if the pile were broken by pressing asunder the plates at any point. Or it may be that the polar condition of the muscular molecules is so delicately balanced as to be disturbed, and, for the time, diminished, by the simple attraction which operates between the sealing-wax or glass *as matter* and the muscular molecules *as matter*.

Under any circumstances, however, there is very insufficient reason for supposing that muscle is stimulated to contract by any kind of *mechanical* agent.

5. *Muscular Contraction in relation to Heat.*—The effects of temperature upon muscular contraction appear at first sight to be at variance with the premises, but they are not so in reality. The plain facts are that muscle will bear considerable variations of temperature without contracting, and that it is thrown into a state of marked contraction, and that equally, by a very high or a very low temperature.

The explanation of this apparent paradox is to be found, not in the immediate effects of the temperature, but in the changes which are wrought by the temperature in the electrical condition of the muscle. So regarding the phenomena, it is quite intelligible that contraction should be caused by low degrees of temperature, for M. Matteucci has shown that the muscular current is suspended under these circumstances. On the other hand, there is every reason to believe that this current is similarly suspended by heat when heat causes contraction. This is certainly the case with regard to the "irritability" of the nerve, as is shown by M. Eckardt in the experiment already cited; and it can scarcely be otherwise with regard to the polar action of both nerve and muscle, for the experiments of M. Du Bois-Reymond go to prove that this action is diminished in every form of muscular contraction. It follows, also, from the same experiments, that the muscular current is not depressed to the point of allowing contraction by any intermediate degrees of temperature; and hence, upon the same premises, it is quite intelligible that the muscle should bear all intermediate degrees of temperature between the extremes without contracting.

Instead of being a paradox, therefore, it is the natural consequence of the workings of temperature upon the muscular current, that the contractions should follow the order which they are found to follow, and being so, it is impossible to say that muscle is stimulated to contract either by heat or by cold.

6. *Muscular Contraction in relation to Light.*—Muscular contraction

appears to be favoured by darkness, and not by light. It is in the darkness certainly, and not in the light, that contraction takes place in the irritable cushions of the sensitive plant; and it appears to be the same with the iris. *It appears to be the same*, for it is more easy to suppose that the iris expands under the stimulus of light, and so closes the pupil, than that this curtain is drawn and the pupil closed by sphincter fibres which have no existence. This explanation is supported by the authority of Bichât; it equally accounts for the phenomena; and it harmonizes with the known influence of light upon the sensitive plant.

7. *Muscular Contraction in relation to Chemical and Analogous Agencies.*—The evidence which belongs to this part of the subject is not so complete as could be desired, but, so far as it goes, it is quite in accordance with the premises. It is furnished by M. Eckandt.* On analysing it, the simple fact is found to be, that the power of inducing contraction which belongs to any of these agents, is directly related to the power which the agent has of destroying the "irritability" of the nerve. The agents themselves act very differently—some by abstracting water from the nerve, some by altering the normal albuminous constituents of the nerve, and some in a more recondite manner: but all destroy the "irritability" of the nerve for the time being, and they do not induce contraction until they have destroyed the "irritability." On experimenting with an acid, for example, the readiness with which contraction may be induced in the muscle by "irritating" the nerve with the point of a needle is found to diminish in direct proportion to the concentration of the acid, and when this concentration is sufficiently great to destroy the "irritability" of the nerve, then, and not till then, is the muscle made to contract by the *acid*. The experiment, in fact, is the precise counterpart of the one related previously, the only difference being that the agency of an acid is substituted for that of heat.

—So far, then, it appears to be altogether improbable that muscle is stimulated to contract by any of the several agencies which have been passed under review: but there are other and more difficult questions which remain in the back-ground, and these must be examined before any definite conclusion can be arrived at.

II.

In this part of the inquiry, the object is to examine into the real nature of muscular contraction, and to point out reasons for supposing that this contraction is nothing more than a passive physical consequence of the common molecular attraction of the muscle.

There are, undoubtedly, many facts which appear to stamp upon muscular contraction the peculiar impress of vitality. How else can the will have any concern in it? Why do the muscles lose so much of their contractile power after death if this power is not a vital endowment? Upon what mere physical hypothesis can the remarkable changes which are exhibited in the *form* of the muscular fibre be accounted for? And if muscular contraction is a vital phenomenon,

* Op. cit., p. 82.

then it is dependent upon stimulus, for life is the stimulus of stimuli.

As to the *will*, it is by no means certain that the action upon muscle is that of a stimulus. Undoubtedly *action* is involved in voluntary muscular contraction, but it is a question whether the *act* be *in the mind*, or *in the muscle*. The will *may act* by withdrawing something from the muscle, as well as by communicating something to the muscle. The will *may act* by suspending the muscular current for the time, and this supposition is in accordance with the premises. At any rate these premises are quite opposed to the idea that the will communicates anything to the muscle during contraction.

There is, undoubtedly, a diminished degree of shortening, and a loss of power after death; but it by no means follows from these facts that the contraction is dependent upon the stimulus of life.

The diminished degree of shortening after death may be nothing more than the simple physical consequence of the circumstances in which the muscle is placed. When a muscle contracts during life, the antagonist muscle either relaxes or opposes no resistance to this contraction. The blood also is fluid, and the intermuscular vessels are readily emptied when pressed upon by the contracting fibres. But after death the spasm is universal, and excess of contraction in any set of muscles is not favoured by the relaxation of antagonist muscles. After death, also, the full degree of muscular contraction may be prevented by the coagulated contents of some of the vessels. It must not be forgotten, however, that muscle can contract to a very great extent after death: thus the ventricular cavities of the heart are frequently obliterated by the contraction of the ventricular walls.

Nor is the loss of muscular strength after death a necessary proof that the contractile power of muscle is a vital endowment. Some loss of strength, indeed, may or rather must, be the natural physical consequence of the circumstances in which the muscular fibre is then placed. In the first place, the fibre may be acted upon by the solvent juices which are present in muscle, and which Liebig has shown to be analogous in their properties to gastric-juice,—these juices acting upon the fibres just as the gastric-juice is sometimes found to act upon the coats of the stomach. Acted upon in this manner, the fibre may be partially dissolved, and to that extent weakened. In the second place, the dead muscle is yielded up to the process of decomposition, and the affinities of the *muscular* molecules may be weakened by the incipient or advanced resolution of these molecules into their constituent elements. Both these causes may combine to produce the result, and, combining, it is evident that the dead muscular fibre must suffer some loss of strength, not because the contractile power of muscle is a vital endowment; but because this power requires for its full manifestation a physical integrity of the muscular fibre which no longer exists.

It is obvious, therefore, that the muscles may lose much of their contractile power after death, without this power being of necessity a vital endowment.

When the movements of the living muscular fibre are considered,

the impression undoubtedly is that these movements are altogether mysterious and peculiar. Why the fibre in contracting should undergo little or no change in bulk, but gain in breadth what it loses in length; and why it should undergo such a remarkable degree of elongation in passing out of the contracted state, appears to be altogether beyond the scope of any physical explanation. The phenomena seem to be too wonderful to be accounted for by anything less than *life*—that mysterious something which, by being more mysterious, is made to account for all mysteries.

On reflecting upon these movements, however, a good deal of their mystery is dispelled, and, in the end, they are found to be capable of receiving a definite physical expression. Muscle, indeed, is made up of fibrin, and this fibrin, for all practical purposes, is identical with the fibrin of the blood. Now, this fibrin of the blood exists in a fluid form, and in a solid form. The fluid form is the living form: the solid, or coagulated form, is that which is assumed on death. Now, the question is, whether or not the fibrin of the muscle undergoes changes which correspond to these. One thing is certain, and this is, that *rigor mortis* is concurrent with the coagulation of the fibrin of the blood. More than this, there is good reason to believe that these two phenomena—*rigor mortis* and *coagulatio mortis*—are not only concurrent, but analogous. What, then, is the condition of the fibrin of the muscle *before rigor mortis?* Is it—like that of the fibrin of the blood—one of fluidity? What is the condition of the fibrin of the muscle in ordinary contraction? Is it, as in *rigor mortis*, one of *coagulation?* These questions naturally arise out of the history of the fibrin of the blood; and if they are answered affirmatively, then there is no difficulty in accounting for the peculiar changes of the muscular fibre.

If, then, the fibrin of the muscle be in a *solid* state during contraction, and in a *fluid* state at other times, it is easy to understand how the fibre may undergo that remarkable change in length which it undergoes when the contraction passes off; for the fluid fibrin will *run* where its course is least impeded, and this is in the direction of the tubes containing it. Again: if the fibrin becomes *solid* in contracting, the form of the contracting fibre need be no cause of difficulty: for this form may be the *natural* form of the fibrin, just as a rhomb may be the natural form of one solid substance, and a cube of another. Nor need there be any change of volume: for many substances solidify without undergoing any such change. Whatever is the real cause of muscular contraction, therefore, there is nothing in the changes of the muscular fibre which *necessitates* the conclusion that these changes are of a vital and mysterious character.

What, then, is muscular contraction? If it is not a vital phenomenon, is it a physical phenomenon? Directly or indirectly everything up to this point has tended to show that it *may* be a physical phenomenon; and there is, indeed, only one serious objection to this conclusion. This arises out of the law of the contraction. If the contraction is the consequence of any known physical attractive force (it is contended), the force of the contraction ought to increase after a definite law as the fibre contracts; but the very reverse is the

actual fact, and the force diminishes as the fibre contracts. Now, there is no doubt that the force diminishes as the fibre contracts; but there is every reason to doubt the correctness of the conclusion which has been drawn from this fact. The experiment of M. Schwann, which is usually cited as the proof, does not warrant any such conclusion. On measuring the force of contraction in the muscles of a frog's leg at different degrees of contraction, M. Schwann found that the force decreases as the muscles contract, and because it does this he concludes that the power cannot be that of molecular attraction. But he curiously forgets that the non-contracting, or imperfectly-contracting cellular substance of the muscle, and the inelastic fluids contained in the muscle, may oppose such a *resistance* to the contraction of the proper muscular fibres, as to mask completely the pure law of that contraction; and doing this his conclusion is altogether invalid. This experiment may indeed show *the degree of resistance* which is opposed to muscular contraction; but it is altogether worthless if it be supposed to show that the law of muscular contraction is essentially different from the law of known physical attractive forces; and it is upon this experiment alone that the idea of this essential difference in the law of muscular contraction is based.

But if muscular contraction is not a vital phenomenon, what is it? Is it the result of an *active* attractive force connected with the state of polar action? There are such attractive forces, unquestionably; but whenever they are present the polar action is also present, and whenever they are increased or diminished the polar action is also increased or diminished. It follows, therefore, that the contractile force of muscle cannot be of this kind, for the current of the muscle fails when this force comes into play, and when the force is manifested permanently, as in *rigor mortis*, the current is for ever extinguished. It follows, also, from the same evidence, that muscular contraction cannot be the result of any *active* physical attractive power, for there are no other forces of this kind besides those which are connected with polar action.

Only one course remains open, therefore, and this is to refer muscular contraction to that common power of attraction which belongs to muscle in common with all matter; for this is the only power which is left after all active powers of attraction are done away with. This is the force which *must* come into play when the muscle ceases to be *resolved* by polar action; and this force is sufficient to account for all the phenomena which yet remain unaccounted for. It accounts for the *power* of muscular contraction, for it is this force, which, acting in the cooling bar of metal, is sufficient to draw in the walls of a bulging building. It accounts, also, for the phenomenon of *rigor mortis*—that phenomenon which is utterly inexplicable on the supposition that muscular contraction is caused by any kind of stimulation; for if this rigor is dependent upon simple molecular attraction, it is quite intelligible that it should come on sooner in cases in which the vitality of the system has been exhausted before death by old age, or by chronic disorder, such as consumption, than in persons who have been cut down suddenly in the full vigour of life, and that the fibre should remain contracted until it breaks up in the ruin of final decay,—for all that is

necessary for the continuance of this contraction is the physical integrity of the fibre. It accounts, that is to say, for those unexplained and seemingly contradictory facts which constitute the distinctive features of *rigor mortis*, and accounting for them, this very circumstance becomes a strong argument that molecular attraction is indeed the cause of muscular contraction.

—The conclusion, then, to which the whole of the previous argument tends is, that muscle is not *stimulated* to contract by any agency, physical or vital, but that contraction is a *passive* phenomenon which *happens* when muscle is left to the play of simple molecular attraction. In other words, *resolution* and not contraction would appear to be the *characteristic* state of living muscle,—this resolution being the natural effect of the muscular current,—and contraction would seem to be nothing more than the return of the muscle to the condition of those tissues which are never relaxed by currents.

III.

In this part it is proposed to show that the special muscular movements which are concerned in carrying on the circulation—"capillary motion" and the rhythm of the heart—can only be explained upon these views of muscular contraction.

1. The manner in which the coats of vessels are affected by the several stimuli which act upon them need be no matter of obscurity. When the nervous energy is exuberant, as in joyous excitement, the skin is flushed; when this energy is depressed, as during fear, the skin is blanched. When the blood is rich and stimulating, as in plethora, the vessels are red and full; when it is poor and watery, as in anæmia, they are shrunk and empty. When the hand is held to the fire it flushes; when exposed to cold it becomes blanched. These phenomena appear to be utterly inconsistent with the idea that the muscular coats of the ordinary vessel are stimulated to contract by nervous influence, by blood or by heat; and there are many phenomena of the kind which are not less inconsistent.

On the contrary, this evidence appears to show that the coats of vessels expand under the influence of these several stimuli, and the test of the correctness of this conclusion is, that this view affords a clue to the interpretation of these mysterious movements of the blood which are independent of the impulse of the heart. In obtaining this clue, it must be assumed, not only that the vessels expand in this way, but that they expand to a far greater extent than the blood which is contained within them, and which is acted upon by the same causes of expansion; and, in order to this assumption, it must be remembered that the dartos and the subcutaneous cellular tissue generally, which tissues are the analogues, or direct representatives of the tissues of which the coats of vessels are mainly built up, are relaxed (expanded) to a very remarkable degree under very small additions of heat, or any other stimuli—a degree to which there is nothing comparable in the blood or in any fluid under any circumstances. Let this be assumed then and the rest is obvious. When stimulated the vessel expands to a greater degree than the blood contained within it, and

the result is that certain vacua would be formed between the vessel and the blood, if more blood did move in to occupy the increased space. Hence, blood must rush into the *stimulated* vessel, and this equally, whether the vessels be acted upon by external heat, as by holding the hand to the fire, or by the natural stimulation of the blood itself within the vessels. In this way the *action* of the blood is to make a way for itself through the vessels.

2. The rhythm of the heart is altogether inexplicable on the supposition that the ventricular systole is the result of stimulation, but upon the opposite theory it is easily disposed of.

The fact that the heart remains distended with blood during a full half of the time occupied in the rhythm is a strong argument that the blood does not excite the ventricular systole; and the history of plethora and anæmia are to the same effect. In plethora the pulse is full and slow; in anæmia empty and quick. In the one case, that is to say, the heart fills to distension with rich blood, and the pulse is deferred; in the other case, the heart takes in a small quantity of poor unstimulating blood, and expels it immediately. The facts are the very opposite of what they would be if the blood excited contraction, for then the pulse would be small and quick in plethora, and full and slow in anæmia. But if the blood provokes the ventricle to expansion by its stimulating properties, then it is intelligible that the heart should dilate more, and the dilatation continue longer when the blood is rich and warm, as in plethora, than when it is poor and watery, as in anæmia.

It may also be presumed that the ventricular systole is not excited by "nervous influence," if any argument may be drawn from what takes place when the nervous energy is more or less depressed, as during fear. Under these circumstances the heart beats hastily, and yet little blood is propelled into the vessels. The beats are perhaps doubled, and yet the skin is cold and pale. Now, under ordinary circumstances, the double number of beats would propel a doubled quantity of blood into the vessels, and the skin would be hot and red, instead of cold and pale; and hence the presumption that, in the apparently anomalous condition of the rapid pulse and pale skin which attend upon fear, the chambers of the heart are diminished in size by the contraction of the walls, and that they thus receive and propel less blood than usual. In other words, the ventricles appear to have contracted *without* nervous influence.

On realizing the actual phenomena of the heart's action, it appears still more improbable that the ventricular systole is caused by stimulation of any kind, and of the blood particularly. At the systole the arterial blood rushes through the coronary arteries into the coats of the heart, and the diastole occurs. The blood remains until it may be supposed to have lost its arterial properties, and then the systole returns. This is the simple fact. It is the *diastole* and not the systole which *appears* to be stimulated by the blood; and this view has the recommendation of affording the key to the rhythm of the heart.

Let it be supposed that the *ventricular* diastole is due partly to the force with which the blood is propelled into the coronary arteries by

the systole, and partly to the stimulation of the arterial blood within the vessels, and (to some extent) within the chambers of the heart. Let it be supposed that this diastole continues as long as the blood retains its arterial properties, and that the systole returns when these properties are exchanged for those of venous blood, and when the stimulus of oxygen is no longer present to avert the systole, and the rhythm is intelligible. Again, the systole restores the diastole; and again, in the same order, systole gives rise to diastole, and diastole to systole, as long as the ventricle can respond to the stimulus of the blood.

It even follows that the *auricular systole* must be contemporaneous with the *ventricular diastole*, for there is good reason to believe that this systole is more the effect of the *falling in* of the auricular walls upon the sudden withdrawal of blood from the auricles by the ventricular diastole, than of any special contraction in the auricles themselves. There is reason to believe this, partly from the absence of valves at the mouths of the veins opening into the auricles, and partly from the structure of the coats of the auricles. If the auricles had to contract *primarily*, it may fairly be assumed that there would have been valves to prevent the reflux of blood into the veins; if they had to contract *rapidly*, it may be assumed with equal propriety that the muscular structure would have been like that of the ventricle or any other muscle which has to contract rapidly, and not, as it is, like that of intestinal or other muscle which is only capable of contracting sluggishly. In this way there is no difficulty in accounting for the movements of the auricles; for the diastole of these organs (which is virtually contemporaneous with the ventricular diastole) is partly due to the same cause—the rush of blood into the coronary arteries—and partly to the onward current of blood which sets in from the veins; and their systole is *mainly* due to the collapse of their walls on the passage of blood into the ventricle, at the ventricular diastole.

Hence, the rhythm of the heart receives a physical explanation, if the blood be supposed to counteract, and not to stimulate, contraction.

The same explanation applies to the movements of the heart, or of a fragment of the heart, after removal from the body. Under these circumstances the air takes the place of the arterial blood, and the only difference is that the cardiac fibres are now stimulated to expand by the oxygen of the air instead of by the oxygen of the blood. If the heart be entire the circumstances are but little changed. The oxygenated air is driven into the coats of the heart through the coronary arteries (partly, at least) by the ventricular systole, and there it causes the diastole; but when this oxygen is replaced by carbonic acid, and the air acquires the negative properties of venous blood, then the diastole must cease, and the systole return. And thus diastole will follow systole, and systole diastole, for some time. Nor is the case very widely different when it is a mere fragment of a heart, which beats rhythmically. Acted upon by the atmosphere, the oxygen excites the muscular current, and induces expansion in the fibre. Fresh supplies of oxygen, however, are required for the continuance of this action; and hence, it follows that the action will fail, and be followed by contraction, when the oxygen in contact with the fibre is converted into carbonic acid. This contraction will dis-

place the old and used-up air, and fresh air will come in to take its place. This fresh air will renew the current, and again place the fibre in the state of expansion, and this expansion will continue so long as the air retains its vivifying properties. Then the resulting contraction will replace the old air with new, and thus expansion will follow contraction, and contraction succeed to expansion time after time. In this way the air will act upon the interior as well as upon the exterior of the fragment, for the action upon the fibres composing the vessels, the cut ends of which are open to the atmosphere, will be to cause these vessels to expand, and to *draw*, as it were, the air into the interior—to draw it in and then expel it, much in the same way, and partly for the same reason, as the air-tubes draw in and expel the air which serves as breath.

There are other arguments of a similar significance to those which have been cited; but sufficient has been said to show that the muscular contraction which is manifested in the coats of the vessels and in the heart, cannot be regarded as the result of stimulation; while, at the same time, the opposite theory is found to give a clue to the explanation of two of the greatest mysteries in physiology, namely, "capillary motion," and the rhythm of the heart.

IV.

In this part—the fourth and last—it is proposed to glance at the pathology of muscular contraction, and show that this is in conformity with the physiological premises.

Now the pathology of the disorders in which muscular contraction is in excess—namely, tremors, convulsions, and spasms, in their multifarious forms—is far too extensive a subject to be considered here, and all that it is possible to do is to glance at the substance of the evidence furnished elsewhere.* This evidence, then, has been elicited from an examination of epilepsy, of affections allied to epilepsy, of the question of periodicity, and of treatment; and this arrangement of the subject had best be preserved here.

1. In epilepsy, then, the condition of the circulation is habitually one of depression. The plethora of the butcher is never met with, and any vascular fulness, if such exists, is mere venous congestion. This depression is aggravated before the fit, and during the fit the condition tends either to syncope or asphyxia. If inflammation, or true fever, chance to be developed, so surely are the convulsions of epilepsy banished for the time. These conclusions are warranted by all the facts of the case.

With this condition of the circulation an active condition of the nervous system is incompatible, and this is quite in accordance with the actual facts. Sense and intellect are completely obliterated during the fit, and at all times they are under a cloud, or if this cloud is occasionally dispelled, and the patient is influenced by any real excitement, he is, for the time, relieved from his fits. *Agitation* may precede the fit, but never true excitement.

The muscles themselves are generally wanting in real tone.

* 'Epilepsy, and other Affections of the Nervous System which are marked by tremor, convulsion, and spasm.' (Churchill, 8vo, 1854.)

The several causes of the malady are all exhausting, not exciting, in their character.

In a word, there is every reason to believe that the muscles of the epileptic contract excessively (as might be expected from the premises), because they are less stimulated than they ought to be, and not for a contrary reason.

2. In affections allied to epilepsy, whether they be marked by tremor, convulsion, or spasm, the same conclusions are arrived at.

The condition of the circulation during the paroxysm invariably tends to syncope or asphyxia, and inflammation or true fever is utterly incompatible with any form of tremor, convulsion, or spasm. Thus, tremor precedes fever, as rigor, and succeeds it, as subsultus; but it never accompanies fever. Thus, convulsion takes the place of rigor or subsultus, but it never happens in the intermediate hot stage of fever. Thus, the spasm of hooping cough disappears if pneumonia or bronchitis are developed, and returns again when the inflammation is over. In every instance the muscular turmoil is coincident with the opposite of vascular activity—the state tending to syncope or asphyxia.

As in epilepsy, so here it may be argued, that this condition of the circulation necessitates a condition of inaction in the function of the nervous system, and this presumption is fully corroborated both by the symptoms during life and the appearances after death. If inflammation of the great nervous centres has been present, the history of the case fully shows that this has been either before or after the tremor, convulsion, or spasm. The patient may be *agitated*, but he is never excited, in the true sense of the word.

The muscles, also, are found, as a rule, to be wanting in tone; and the so-called *exciting* causes are always *depressing* in their character.

Everything, indeed, tends to support the previous conclusions, and to show that in affections allied to epilepsy, as in epilepsy itself, and in ordinary muscular contraction, the muscles contract independently of any increased stimulation. The physiology explains the pathology, and the pathology confirms the physiology.

3. The phenomena of periodicity also point to conclusions of the same kind. The plant exhibits plainer and more numerous evidences of periodicity than the animal, and it does this, it is argued, because it has less of that innate life which enables the higher animals to be partially independent of the vivifying influences which are derived from the outer world. If man exhibits more evidences of periodicity than he ought to do, it follows, therefore, that he is shorn of some of that innate life which is the badge of distinction between him and the plant; and hence the periodicity of epilepsy or any cognate disorder is merely one proof that the system in which these diseases are manifested is less stimulated—less vitalized than it ought to be.

4. If, then, these diseases are of this character, it follows, as a necessary consequence—what, indeed, may almost be said to be proved by experience,—that bleeding, purging, or any lowering measures are not calculated to do any good; and that the only hope of benefit must be placed upon measures which will not only strengthen, but *rouse* the system,—a conclusion which is fully warranted by the experience of the author.

— The only conclusion, then, which can be drawn from the consideration of those special muscular movements which are manifested in the coats of vessels, and of the pathology of the subject, is the same as that already drawn ; and the final conclusion must be, *that muscle does not contract in consequence of the communication of any stimuli to the muscle, but that it contracts because the common molecular attraction of the muscular fibre is no longer counteracted by the action of these stimuli upon the muscle.*

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